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Blood Lactate Removal During Varying Intensities of Active Recovery Following Supramaximal Work.

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**BLOOD LACTATE REMOVAL DURING VARYING INTENSITIES OF
ACTIVE RECOVERY FOLLOWING SUPRAMAXIMAL WORK**

The Louisiana State University and Agricultural and Mechanical Col. **PH.D.** 1982

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BLOOD LACTATE REMOVAL DURING VARYING
INTENSITIES OF ACTIVE RECOVERY
FOLLOWING SUPRAMAXIMAL WORK

A Dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy

in

The School of Health, Physical Education,
Recreation and Dance

by

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Dedicated to
my mother and father
for all the years
of encouragement, patience
and, most of all, love

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ABSTRACT

The purpose of this study was to investigate the effect of varying intensities of recovery exercise on the level of blood lactate after a short, supramaximal exercise bout. Seven males were advised of the testing protocol and consented to serve as subjects.

All subjects performed an oxygen cost of cycling test and maximal oxygen uptake ($\dot{V}O_2$ max) test prior to the commencement of the recovery tests. The subjects then performed one of four treatments on separate days with 1-4 days of rest between tests. Preceding each treatment the subjects cycled at a workload corresponding to 150% of $\dot{V}O_2$ max for 50 secs. At the end of this work bout the subjects performed one of the following treatments for 40 min: (1) passive recovery, (2) cycling at 35% of $\dot{V}O_2$ max, (3) cycling at 65% of $\dot{V}O_2$ max, (4) cycling at 65% for 7 min followed by cycling at 35% for 33 min. In addition, the same recovery treatments were performed on separate days without the prior supramaximal bout to determine baseline lactate values. These values could then be used in comparing the recovery rates.

Serial blood samples were obtained throughout the recovery exercise to determine lactate values with one venepuncture sample taken at 20 min for the baseline

value. The blood samples were analyzed by an enzymatic technique.

The peak in blood lactate occurred at the same time for each treatment with no significant difference ($p > 0.05$) in the absolute lactate value. The rate of removal of lactate showed no significant differences ($p > 0.05$) between the combination recovery and the 35% recovery. The absolute value of lactate at the end of the recovery period exhibited no significant differences ($p > 0.05$) between the 35% and combination recoveries. However, the absolute value of the 65% recovery at the end of 40 min was greater than that found in the passive recovery.

These data indicate that a single intensity recovery that increases blood flow and muscle metabolism without causing significant lactate production may be the optimal method of removing blood lactate.

CHAPTER I

INTRODUCTION

It is generally agreed that lactate production and accumulation is a cause of fatigue and an inhibitor to muscle contraction during short-term, maximal work (Fitts & Holloszy, 1976; Karlsson, Bonde-Petersen, Henrikkson, & Knuttgen, 1975; Klausen, Knuttgen, & Forster, 1972; Stamford, Rowland, & Moffatt, 1978). In sports such as track, cycling and swimming, some athletes compete in more than one event during the course of a competition. In events such as long sprints and middle distances (i.e., track events such as 400 and 800 meters), where the energy supply is produced primarily from glycolytic metabolism, the accumulation of cellular lactate can be substantial. In many circumstances the athletes may have 30-60 min to recover prior to the next event and normally rest in a sitting or supine position. But, is this the optimal recovery method to lower blood lactate levels? Jervell (1928) showed that blood lactate concentration could be made to fall faster, compared with resting conditions, if moderate exercise was performed in the recovery period. However, the intensity of exercise performed during recovery to optimize blood lactate removal is not clear (Belcastro & Bonen, 1975; Hermansen & Stensvold, 1972). Recently, Stamford, Weltman,

Moffatt and Sady (1981) have suggested that the optimum method of blood lactate removal may not be continuous exercise at a constant workload but may involve recovery exercise at several loads. However, at present, no attempt has been made to examine a combination of exercise intensities to remove lactate from the blood following maximal work. Therefore, the purpose of this study was to compare the rates of blood lactate removal following maximum exercise using varying intensities of recovery. The recovery treatments were passive recovery, exercise at 65% of $\dot{V}O_2$ max, exercise at 35% of $\dot{V}O_2$ max, and exercise at a combination of 65-35% of $\dot{V}O_2$ max.

REVIEW OF LITERATURE

Lactate Production in Muscle

Biochemical Considerations. Since stored ATP in muscle will produce energy for only a few seconds, it must constantly be resynthesized. When sufficient substrates, oxygen and oxidative enzymes are present, this process takes place primarily through oxidative phosphorylation. If the substrate, oxygen or oxidative enzyme supply is deficient, the ATP is produced primarily in the glycolytic pathway. During periods of accelerated glycolysis, the rate of production of pyruvate exceeds the rate of oxidation of pyruvate by the citric acid cycle. Also, under anaerobic conditions more reduced nicotinamide adenine dinucleotide

(NADH) is formed than is oxidized in the respiratory chain. This inability of pyruvate to be oxidized may be caused by the buildup of NADH in the electron transport system or by an insufficient oxygen supply in the tissue as in heavy exercise (Gollnick & King, 1969) or during exercise at high altitude (Hermansen & Saltin, 1967). On the other hand, it may be caused by an inadequate supply of oxidative enzymes in the muscle (Keul, Doll, & Kuppler, 1967). For glycolysis to continue during anaerobic metabolism, oxidized nicotinamide adenine dinucleotide (NAD⁺) must be available for oxidation of glyceraldehyde 3-phosphate. This is accomplished by the enzyme lactate dehydrogenase (LDH) which oxidizes NADH to NAD⁺ and reduces pyruvate to lactate. Thus, the formation of lactate allows glycolysis to continue longer than possible if it were not formed (Stryer, 1981).

Muscle Fiber Types. The inter-conversions of pyruvate and lactate are mediated by different properties of the enzyme lactate dehydrogenase (LDH) found in skeletal muscle and liver. LDH has two kinds of polypeptide chains called muscle (M) and heart (H), which can form five types of tetramers: M₄, M₃H₁, M₂H₂, M₁H₃, and H₄. The M₄ isozyme has a much higher affinity for pyruvate than does H₄, while the other isozymes have affinities lying somewhere between these two. The isozyme found

mainly in skeletal muscle is M4 with H4 being the main isozyme in the liver and heart (Stryer, 1981). However, any of these tissues may have some of each type of LDH. Skeletal muscle with high aerobic capacity has been found to have a predominance of the H-type isozymes (Dawson, Goodfriend, & Kaplan, 1964). Therefore, these fibers have a greater tendency to convert pyruvate to acetyl Co-A instead of lactate. The fast twitch fibers, on the other hand, have mostly M4 isozymes and produce greater amounts of lactate.

The manner in which energy is produced also depends on the type of muscle fiber. Basically, muscle is made up of red and white fibers. The white fibers, with high levels of glycolytic enzymes, function during exercise primarily by resynthesizing ATP through anaerobic glycolysis. The red fibers, with high levels of oxidative enzymes, are adapted to resynthesize ATP at rest and during exercise by aerobic means using primarily blood borne substrates (Keul et al., 1967). Therefore, it is the fast twitch fibers that are generally thought to be responsible for much of the lactate production in the muscle during work.

Lactate Production During Exercise. Before the mechanism of lactate production can be examined, the methods by which it has been measured should be considered.

The measurement of lactate production has been accomplished by several techniques. Analysis of the arterio-venous concentration differences within a group of muscles or by measuring arterial concentration variation has been the most common (Issekutz, Shaw, & Issekutz, 1976; Jorfeldt, Juhlin-Dannefelt, & Karlsson, 1978; Stainsby & Welch, 1966). Lactate formation in muscle has also been studied by analysis of the lactate content in muscle tissue collected by needle biopsy technique immediately after exercise (Diamont, Karlsson, & Saltin, 1968).

There is considerable evidence that lactate production is linked to a deficient oxygen supply (Bannister & Cunningham, 1954; Karlsson, 1971b; Pentecost, Reid & Reid, 1966). The question must be raised as to why does resting muscle produce lactate? Also, why is lactate produced at steady-state submaximal work and why is venous PO_2 only slightly changed? Bang (1936) found that, with the onset of exercise, an increase in plasma lactate is seen. It is generally agreed that the blood levels rise in an exponential manner with increasing work intensity (Bang, 1936; Hermansen & Saltin, 1967). Bang also found that the blood lactate concentration during prolonged, submaximal exercise, peaked about 5 min into the work and then declined to the pre-exercise level. Contrarily, Genolevy (1980) and Stamford et al. (1981)

found that blood lactate accumulation remains steady at an elevated level from 20 to 60 min of submaximal exercise. Neither study delineates the mechanism by which the lactate increases. This contrast has created two schools of thought on the production of lactate. Is lactate produced constantly or just at the beginning of exercise and stored in inactive tissue to be released later?

Bang's conclusion was that lactate was produced only in the first few minutes of exercise and corresponded to the oxygen deficit. Hughes, Clode, Edwards, Goodwin, and Jones (1968), Saiki, Margaria, and Cuttica (1967), and Stainsby and Welch (1966) support Bang's hypothesis that lactate produced is limited to the first phase of exercise before oxygen consumption has reached a steady state. However, at high work intensities, at or above the anaerobic threshold, the lactate concentration does not decline (Bang, 1936; Hermansen & Stensvold, 1972). Therefore, the observed concentration of blood lactate indicates only the net result of addition and removal of blood lactate so that the steady elevated concentration during exercise might represent lactate produced at the beginning of work and not metabolized or removed until recovery. The lactate produced could be held by inactive muscle and released later. This is supported by Poortmans, Bossche, and Leclercq (1978) who

found that approximately 18% of the lactate entering inactive muscle is metabolized.

But, why then, if lactate is constantly being metabolized, as demonstrated by Jorfeldt (1970) and Hubbard (1973), does the blood concentration not fall over 60 min duration of submaximal work? This leads to the alternate conclusion that there is a dynamic equilibrium between continuous addition to and removal from the circulation. Hubbard (1973) and Jorfeldt (1970) provide the only data to support this continuous production and uptake. This could be explained by the muscle having different fiber types (Hubbard, 1973; Jorfeldt, 1970). With the muscle having a mixture of fiber types, the white fibers could produce lactate at the same time that the red fibers were metabolizing lactate (Issekutz et al., 1976).

In summary, there is no definitive answer to the question of whether lactate is produced constantly or transiently during submaximal work. Perhaps future work using in situ muscle preparations will increase our understanding of this important question.

Lactate Efflux

Jorfeldt (1970), in studying lactate release at submaximal workloads, found that the release was lower after 40 min of work than after 10 min. Then, using the same protocol, he injected radioactive lactate into the subjects and found the release was lower

at both time periods. The labelled CO_2 which was measured after injection exhibited a biexponential curve which suggests that some is stored in pools with different diffusion rates. Therefore, the blood lactate concentration may only represent the metabolic events of the muscle in a qualitative manner.

Jorfeldt et al. (1978), in studying this translocation problem, found that the release of lactate failed to keep up with its formation at muscle concentrations above $4 \text{ mmol} \times \text{kg}^{-1}$ wet muscle weight. This could not be explained by blood flow since both blood flow and oxygen consumption increase linearly with work loads up to 90% of $\dot{V}\text{O}_2$ max. He hypothesized that this could be because of active processes in the cell membrane or by extracellular determinants such as an imbalance between recruited fibers and available draining capillaries. Thus, it seems as though the diffusion of lactate from the cell to the blood takes place by passive diffusion up to some point around $4 \text{ mmol} \times \text{kg}^{-1}$ wet weight at which time either the cell membrane or the extra-cellular components are changed to cause the diffusion to become altered. On the other hand, it could be an active process in which the carrier's capacity is sufficient up to a blood lactate concentration of approximately 4 mmol , at which point it is saturated and transport is hampered. Thus, the process of

translocation of lactate is a confounding factor when trying to interpret blood lactate values.

Blood Lactate Removal

Blood lactate removal and metabolism depend on a complex interaction of several factors. The primary factors include (1) muscle fiber type, (2) the ratio of active to inactive skeletal muscle, (3) hepatic uptake of lactate, and (4) concentration of lactate in the blood (Stamford et al., 1981).

Bar and Blanchaer (1965) found lactate uptake to be greater in red muscle fibers, when compared to white fibers, probably because the LDH was of the H-type. Supporting this, Sjodin (1976) found the red or slow-twitch fibers to contain approximately twice as much H-LDH as M-LDH. Also, the relationship between lactate uptake and the percentage of slow-twitch fibers may be related to the metabolic and anatomical features of the fibers. The slow-twitch fibers also have a greater surface area which may allow greater uptake (Anderson, 1975).

Margaria, Edwards and Dill (1933) and Poortmans et al. (1978) found that non-exercising muscles could also extract and metabolize lactate. However, Poortmans et al. (1978) revealed that only 18% of the extracted amount was metabolized. Thus, the active skeletal muscles are responsible for metabolizing a much greater amount of lactate. Stainsby and Welch (1966) and

Jorfeldt (1970) found that exercising muscle could indeed extract lactate at high arterial concentrations. The heart also extracts lactate for metabolism even at normal levels but increases its extraction, as do the skeletal muscles, at high arterial concentrations (Goodale, Olsen, & Hackel, 1959).

Another important factor affecting removal is hepatic uptake of lactate. Hepatic blood flow during mild (heart rate $130 \text{ beats} \cdot \text{min}^{-1}$) and strenuous exercise (heart rate $180 \text{ beats} \cdot \text{min}^{-1}$) decreases to approximately 80 and 50% of resting values, respectively (Rowell, Blackmon, & Bruce, 1964; Rowell, Kraning, Evans, Kennedy, Blackmon, & Kusumi, 1966). However, the hepatic arterio-venous difference of lactate is increased which suggests that removal is still effectively taking place (Rowell, 1971). Rowell (1971) also found that this lactate removal by the liver is the same during exercise as it is at rest. Brooks, Brauner & Cassens (1973) found that, in animals, the hepatic glycogen synthesis from lactate is minimal. In support of this, Depocas, Minaire, and Chatonnet (1969) found that approximately 10% of the lactate in the blood is converted to glucose at rest or in exercise.

Bonen, Campbell, Kirby and Belcastro (1979) have suggested that blood lactate removal is most closely associated with intensity of recovery, slow-twitch

muscle fiber content, and blood lactate concentration. In order to test the effects of high concentrations of blood lactate on tissue uptake, Jorfeldt (1970) injected labelled lactate into human subjects and found the uptake to be lower at 10 and 40 min of exercise than during exercise at normal concentrations. He also found 30-50% of the uptake of labelled lactate in the form of labelled carbon dioxide and 20% as other metabolites. This leaves approximately 30% of the labelled lactate which must be accounted for by the incorporation of labelled carbon dioxide into metabolic pools with slow turnover rates. Jorfeldt (1970) also found that the rate of lactate uptake was best correlated with lactate inflow to the muscle (blood flow X concentration). This rate of uptake exhibited a curvilinear relationship that suggests the existence of saturation kinetics. It is unclear whether this is due to (1) a limited rate of transport of lactate across the cell membrane or (2) a limited rate of metabolism of the extracted lactate.

Hubbard (1973) showed that labelled lactate injected intravenously decreased more rapidly during exercise than at rest. This was attributed to an increased rate of metabolism as evidenced by a more rapid elimination of labelled carbon dioxide in expired air. During exercise, 35-68% of the radioactivity administered as lactate was recovered as expired labelled carbon

dioxide compared to 3-7% at rest. A small proportion of the radioactivity in blood was derived from the conversion of lactate to glucose with the urinary excretion of labelled lactate being negligible up to 9 hours post injection. This is in agreement with Jorfeldt (1970) in that he found 30-50% of the labelled lactate injected into the brachial artery during forearm exercise was immediately oxidized and recovered as labelled carbon dioxide. Since gluconeogenesis is not considered to take place in the skeletal muscle (Krebs, 1964), the oxidation of lactate via pyruvate and the tricarboxylic acid cycle is the probable route of lactate metabolism in muscle. This utilization of lactate by active muscle is supported by Stainsby and Welch (1966) and Karlsson (1971a).

Metabolism of Blood Lactate

Hill and Lupton (1923) proposed that the so-called "lactic acid" portion of the oxygen debt was caused by the oxidation of one-fifth of the lactate to provide energy to convert the other four-fifths to glycogen. Contrarily, Brooks et al. (1973) found that lactate was not used for glycogen synthesis. He found that 70-90% of labelled lactate appeared as carbon dioxide. He felt that the increase in catecholamines during exercise, with their enhancement of glycogen degradation, seems to support these findings of no glycogen synthesis. This leaves two

possibilities for the oxidation of the lactate:

(1) conversion of lactate to pyruvate which is oxidized via the Krebs cycle or (2) lactate is taken up by the liver and converted to glucose.

Gisolfi, Robinson and Turrell (1966) and Bonen et al. (1979) hypothesized that lactate being utilized as fuel is the preferred pathway causing the reduction in glycogen synthesis. Normally, during light exercise in the range of 20-50% of $\dot{V}O_2$ max, the major portion of energy metabolism would be fueled by free fatty acids (FFA) (Ahlborg, Hagenfeldt, & Wahren, 1976). However, during periods of high lactate concentration in the blood, FFA mobilization might be expected to be inhibited (Issekutz & Miller, 1962). Yet, there is also evidence that lactate promotes the uptake of FFA in muscle (Ahlborg, et al., 1976; Dieterle, Banholzer, Dieterle, Henner, & Schwartz, 1971). In spite of the conflicting evidence, it seems reasonable that the lactate is a major source of fuel for oxidative metabolism during active recovery exercise. In support, Depocas et al. (1969) found 74% of lactate formed was promptly converted to CO_2 and that 10-20% of plasma glucose is derived from lactate at rest and exercise. The glucose is present because both lactate and pyruvate diffuse out of active skeletal muscle into the blood and are carried to the liver. Generally, more lactate than

pyruvate diffuses out of the cell because of the high NADH/NAD⁺ ratio in contracting skeletal muscle. Lactate entering the liver is oxidized to pyruvate because of the low NADH/NAD⁺ ratio in the cytosol of the liver. Pyruvate may then be converted to glucose in the gluconeogenic pathway of the liver. Glucose then diffuses to the blood and may be taken up by skeletal muscles and/or other tissues. This reconversion is called the Cori cycle and is important in understanding production and uptake of lactate (Stryer, 1981). Other estimates of glucose recycling in man also show 20% of glucose turnover participates in this cycle (Kreisberg, Siegal, & Owen, 1971; Reichard, Moury, & Hochella, 1963; Waterhouse & Keilson, 1969). This resynthesis requires six molecules of ATP which are provided by fat oxidation in the liver (Kreisberg, 1972). Issekutz et al. (1976) found approximately the same fraction of lactate conversion to glucose at rest as during exercise in the dog, 19 and 25% of the lactate turnover rate, respectively. He also found that at rest and during exercise the oxidation of lactate was 50-55% of the turnover rate. In addition, at rest, 9% of hepatic glucose was derived from lactate and during exercise this increased to as much as 25%.

Thus, as can be seen, approximately 90% of lactate produced can be accounted for by immediate

metabolism or conversion to glucose. This suggests that, although much of the lactate is metabolized, the lactate which is unaccounted for immediately may be stored and metabolized later. If this storage does indeed take place, it may help explain the contradicting data concerning lactate production.

Effects of Exercise on Blood Lactate Removal

Over 50 years ago it was shown that blood lactate concentration could be made to fall faster during light, submaximal work than during rest (Hill & Lupton, 1923; Jervell, 1928). This was later examined and verified by many other investigators (Bang, 1936; Newman, Dill, Edwards, & Webster, 1937; Gisolfi et al., 1966; Rowell et al., 1966; and Davies, Knibbs, & Musgrove, 1970). However, the relationship between the rate of lactate removal and the severity of the work load performed in the recovery period is not yet clear (Belcastro & Bonen, 1975; Hermansen & Saltin, 1967; Stamford, Moffatt, Weltman, Maldonado, & Curtis, 1978).

Several factors previously mentioned affect the rate of lactate removal at rest. Bonen et al. (1979), in attempting to determine which of these factors were the most important during exercise, developed a multiple regression model to predict

lactate removal rates after exhaustive exercise. Their subjects cycled at recovery rates ranging from 29 to 49% of their $\dot{V}O_2$ max. Their model employed three independent variables: (1) slow-twitch fiber content of muscle, (2) the lactate concentration in the blood, and (3) the intensity of the recovery exercise. The authors also found that, for resting recovery, only the peak lactate concentration provided valid data for prediction of the rate of lactate removal. They also found a significant correlation between the blood lactate concentration and the lactate removal rate during an active recovery period which suggests that blood lactate also exerts a mass action effect in man. This trend was also seen at rest in subjects exhibiting an acceptable linear decrease in lactate removal.

The peak lactate removal rate seems to be between $7-8 \text{ mg} \cdot \text{dl}^{-1} \cdot \text{min}^{-1}$ at treadmill intensities of 55-70% of $\dot{V}O_2$ max (Hermansen & Stensvold, 1972; Belcastro & Bonen, 1975). This rate was calculated by determining the slope of the linear portion of plasma lactate concentration curve during recovery.

Stamford et al. (1978) found that breathing 100% oxygen during one-leg cycling as a recovery from a near maximal exercise bout of 5 min was no more beneficial than one-legged recovery breathing a normoxic gas. This could be explained by the fact that breathing

hyperoxic gas decreases muscle blood flow (Welch, Bonde-Peterson, Graham, Klausen, & Secher, 1977). In support Weltman, Stamford, and Fulco (1979) found recovery above anaerobic threshold while breathing 100% oxygen to be no more effective in removing blood lactate than recovery at approximately 40% $\dot{V}O_2$ max breathing normoxic gas.

The mode of exercise performed during recovery has been shown to affect the rate of blood lactate removal. For the treadmill, several investigators have shown that the optimal recovery intensity for blood lactate removal is somewhere between 60-80% of $\dot{V}O_2$ max (Belcastro & Bonen, 1975; Gisolfi et al., 1966; Hermansen & Stensvold, 1972). Since splanchnic blood flow was probably reduced at these intensities, lactate uptake by skeletal muscles and the heart probably compensated for the reduced transport of lactate to the liver. Also, Hermansen and Saltin (1967) have shown that well-trained subjects have lower blood lactate concentrations than untrained subjects at the same absolute or relative workloads. It is assumed that the intensity was below the anaerobic threshold and thus no additional lactate was being produced. A poorly conditioned subject probably would have been producing a significant amount of lactate at that intensity and thus blood levels of lactate would be increasing. In support of these findings, Bonen and Belcastro (1976) found that a self-selected jogging pace,

later found to correspond to about 65% of $\dot{V}O_2$ max, was preferential over rest or a series of intermittent activities (calisthenics, walking, or jogging).

Previous data have shown that cycle ergometer exercise recovery intensity is optimal between 25 and 40% of $\dot{V}O_2$ max for the removal of blood lactate (Belcastro & Bonen, 1975; Davies et al., 1970; Dyksta, Boileau, & Misner, 1973). These studies have also shown that recovery intensities of 60-80% of $\dot{V}O_2$ max on the cycle are no more advantageous than resting recovery. In testing four different intensities, Bonen and Belcastro (1976) found that the optimal rate for lactate removal was 32% of $\dot{V}O_2$ max. They also found that the subjects, when given the opportunity to select their own recovery intensity, selected one that was as effective in removing blood lactate as the 32% of $\dot{V}O_2$ max recovery.

Stamford et al. (1978) used the cycle to test the hypothesis that non-fatigued working muscles may be more efficient in extracting lactate from the blood than fatigued muscles. This was accomplished with one-legged cycling to exhaustion followed by active recovery with the opposite leg. Further, they examined resting recovery and using the exercising leg at an even lower intensity. It was found that the recovery with the reduced load on the exercising leg was the most advantageous in removing lactate. The factor most closely

associated with the faster removal of lactate was the predetermined baseline accumulations of lactate for the various treatments. The baseline values were determined by performing the recovery treatment without the preceding fatiguing exercise. If the baseline lactate values were not significantly different between two treatments, blood flow and percent of $\dot{V}O_2$ max were the major determinants of lactate removal rates. Contrarily, if baseline blood lactate values were significantly different and blood flow and percent of $\dot{V}O_2$ max were the same between two treatments, the treatment with the lower baseline lactate value has the fastest lactate removal rate. These findings were consistent with others in that these optimal recovery rates were approximately 30-35% of $\dot{V}O_2$ max.

This raises the question of using the previously mentioned baseline determinations for each intensity to examine the rate constants of the decreasing lactate concentrations. This was done by Stamford et al. (1981) with 70% and 40% of $\dot{V}O_2$ max and resting recovery. They found no difference in the lactate removal rate constant between the 40% and 70% recovery intensities when comparing them to predetermined baselines. However, blood lactate removal was greater at both 70% and 40% $\dot{V}O_2$ max than during resting recovery. If the resting baseline is used, the 40% recovery had a faster half time than the resting or the 70% recovery.

The reason for the higher optimal recovery intensities on the treadmill as opposed to the cycle may be because of the amount of muscle mass used to do the work. Freyschuss and Strandell (1967) have shown that the same relative work load distributed over a larger muscle mass results in a lower lactate production. This is supported by the work of Asmussen and Neilsen (1946) and Stenberg, Astrand, Ekblom, Royce, and Saltin (1967). Again, the fitness level of the subject also has an influence on lactate production and removal at any given work rate.

In summary, many unanswered questions remain in describing the relationship between exercise and blood lactate disappearance. Several factors interact and affect blood lactate removal and, as yet, one variable cannot be singled out to determine the importance or timeliness of each. Stamford et al. (1981) has suggested that a relatively high recovery workload may cause blood lactate levels to peak earlier following maximal exercise and decline at a faster rate during the first 5-7 min of work than a lower relative workload. In contrast, the lower relative workload will cause a faster rate of decline in the recovery period after 5-7 min. Thus, one single recovery intensity may not be optimal. By using a combination of intensities to examine the rate of lactate removed, the factors that affect removal, as well as the optimal intensities, may be determined.

Statement of the Problem

The purposes of this study were to:

1. Determine if there are significant differences among the rates of blood lactate removal while at rest, cycling at 35% of $\dot{V}O_2$ max, at 65% of $\dot{V}O_2$ max, and at a combination of 65 and 35% of $\dot{V}O_2$ max after a 50 sec work bout at 150% of $\dot{V}O_2$ max.
2. Determine if significant differences exist among the absolute values of blood lactate at peak values or from mins 20 to 40 during the recovery at rest, cycling at 35% of $\dot{V}O_2$ max, at 65% of $\dot{V}O_2$ max, and at a combination of 65 and 35% of $\dot{V}O_2$ max after a 50 sec work bout at 150% of $\dot{V}O_2$ max.
3. Determine if significant differences exist among the rate constants of the curves describing the blood lactate removal when using the baseline corrections as compared to a resting baseline.

Research Hypotheses

1. There will be significant differences ($p < 0.05$) in lactate removal rates among all four treatments across time. The optimal lactate removal rate will be seen in the combination recovery followed by the 35% recovery, the 65% recovery, and the passive recovery.
2. There will be significant differences ($p < 0.05$) in the absolute lactate values among all four treatments from 20 to 40 min of recovery. The 65-35% combination

will have the lowest lactate value followed by the 35%, 65%, and resting recoveries.

3. There will be significant differences ($p < .05$) in lactate removal rates among all four treatments when compared to predetermined baselines. The 65-35% combination will have the optimal rate followed by the 35%, 65%, and the passive recoveries.

Operational Definitions

Steady state. A submaximal exercise condition during which the oxygen uptake, as well as other physiological variables such as ventilation, heart rate, cardiac output, etc., are relatively constant.

CM-5 lead placement. Electrodes placed over the sterno-clavicular joint and on the right and left sides in the fifth intercostal space at the anterior axillary line.

Lactate. Preferred term over lactic acid since the pK_a for lactic acid is more than 3 pH units lower than physiological pH. Thus, at physiological pH, lactic acid releases a hydrogen ion and forms the salt lactate.

Assumptions

1. If the hand is kept warm, the blood sample taken from a vein on the lower forearm is not significantly different in lactate concentration from an arterial sample.

2. The recovery exercise at approximately 35% of $\dot{V}O_2$ max does not increase the accumulation of blood lactate significantly over resting values.

3. The recovery exercise at approximately 65% of $\dot{V}O_2$ max is above the anaerobic threshold of the subjects and will produce significantly more lactate than the other treatments.

Limitations

1. Because of the invasive technique involved, examining the arterio-venous difference in blood lactate is not feasible. Measurement of blood lactate reflects the relationship between lactate addition to the blood and lactate removal from the blood. Thus, the absolute rate of blood lactate removal cannot be quantified from analysis of venous blood samples alone.

2. Since the fiber type ratio of the subjects is unknown, it is only possible to choose subjects that perform similar activities and are close to the same fitness levels.

Significance

Not only does the literature show inconsistencies in lactate removal when comparing work on the cycle and treadmill, it also reports discrepancies when comparing work on the same instrument. These inconsistencies may be caused by factors such as using different modes of work to produce high levels of blood lactate and

therefore creating various peak levels of lactate in the blood. If each subject had a different peak in blood lactate, because concentration is a determinant of removal rate, the rate would be influenced and the effect of the recovery intensity would be masked. Therefore, an attempt to create similar peaks in blood lactate for each subject would allow comparison of recovery intensities not yet undertaken.

Also, since Stamford et al. (1981) have shown recovery at an intensity above the anaerobic threshold creates an earlier peak in blood lactate values, the combination recovery could elicit a quicker recovery. This concept is supported by the data of Graham and Sinclair (1978) in which they concluded that the major determinant of lactate removal from the blood is blood flow to the working muscles. This also would be accomplished during the first phase of the combination recovery. Therefore, using a combination of recovery intensities, while helping to determine an optimum rate of blood lactate removal, may also delineate more clearly those factors involved in the mechanisms of lactate production, diffusion, and uptake.

CHAPTER II

Methods

Subjects

The study utilized seven male volunteers as subjects who trained primarily through jogging or cycling. The subjects were informed of the nature of the study before the commencement of any tests and asked to sign a consent form in accordance with the Human Use Committee at Louisiana State University (Appendix A). Biometric data on the subjects are presented in Table 1.

Procedures

Oxygen Cost of Cycling and Maximal Oxygen Uptake.

The subjects arrived at the laboratory 4 hours post-prandial and performed a warm-up of 50 watts (60 rpm) for 2 min duration on a cycle ergometer (Monark). After 2 min rest, the subject performed an oxygen cost of cycling test to determine the load at which a given $\dot{V}O_2$ could be attained. The test consisted of three separate 6-min work bouts at work loads of 50, 100, and 150 watts (60 rpm) with a 10-min recovery period between loads. Oxygen consumption was measured from the 4th to the 6th minute of exercise. Upon completion of the oxygen cost of cycling test, the subject then rested for 15 min prior to the $\dot{V}O_2$ max test. The max test began at a workload of 100 watts and

Table 1
Biometric Data of All Subjects

| Subject | Age (yrs) | Height (cm) | Weight (kg) | $\dot{V}O_2$ max (ml/kg x min ⁻¹) |
|----------|--------------|----------------|----------------|--|
| 1 | 32 | 185.0 | 84.0 | 45.3 |
| 2 | 26 | 172.7 | 59.0 | 55.9 |
| 3 | 26 | 170.0 | 75.0 | 44.9 |
| 4 | 40 | 180.3 | 76.8 | 46.8 |
| 5 | 27 | 170.2 | 70.0 | 51.0 |
| 6 | 28 | 172.7 | 78.4 | 49.3 |
| 7 | 29 | 181.8 | 69.5 | 47.7 |
| <hr/> | | | | |
| X ± S.D. | 29.7 ± 4.9 | 176.1 ± 6.1 | 73.2 ± 8 | 48.7 ± 3.7 |

increased 25 watts per minute until the subject was unable to continue the test. Heart rate (HR) and ventilation (\dot{V}_E) were recorded each minute with expired gas fractions being recorded every 10 sec throughout the test. Oxygen uptake was calculated using the Haldane transformation of the Fick equation. The criterion of a plateau or a drop in oxygen uptake with an increased work load, along with examination of respiratory quotient (RQ) and heart rate (HR), were used to determine if the $\dot{V}O_2$ max was attained (Astrand & Saltin, 1961a; Astrand & Saltin, 1961b). If the subject did not meet the listed criteria, the test was repeated until the above criteria were met.

Recovery Tests. Prior to each recovery test a short, supramaximal exercise bout was administered to each subject to increase blood lactate substantially. The bout, performed on the cycle ergometer, was of 50-sec duration to assure that the peak blood lactate concentration would occur after the exercise bout was terminated and the recovery period had begun. The subject pedalled with no load for 10 sec in order to attain a constant pedalling frequency (80 rpm). After 10 sec of pedalling, the load was increased to 150% of the subject's $\dot{V}O_2$ max over a 10-sec period and the subject continued to work at this rate for 50 sec. Immediately at the end of this supramaximal work bout,

the desired recovery load was set and the subject continued pedaling for 40 min at one of the three loads or rested quietly. One to four days were allowed for rest between each treatment.

The four recovery conditions were (1) passive recovery (seated), (2) 65% of $\dot{V}O_2$ max, (3) 35% of $\dot{V}O_2$ max, and (4) a combination of 65 and 35% of $\dot{V}O_2$ max (combination recovery). During the passive recovery the subjects sat in a chair placed next to the cycle for 40 min. During the 65% and 35% recoveries the subjects continued to cycle at 60 rpm at a predetermined load which elicited the desired relative workload for the 40-min period. For the combination recovery period the initial load of 65% of $\dot{V}O_2$ max was utilized for 7 min at which time the load was adjusted to required 35% of $\dot{V}O_2$ max for the remaining 33 min. This combination was chosen to create the early peak in lactate reported to be seen with a high work load along with the faster removal rate seen with the lower work load. The recovery intensity of 65% of $\dot{V}O_2$ max was chosen to keep blood flow high to the working muscle and thus compensate for the decreased hepatic lactate removal. The 35% intensity was chosen because the consensus of opinion among previous investigators places the optimal removal rate for the cycle ergometer at this intensity, one that results in no significant lactate production or

reduced hepatic blood flow. Samples of blood for determination of lactate concentration were collected at rest and at 1, 2, 3, 4, 5, 6, 7, 9, 12, 15, 20, 25, 30, 35, and 40 min of recovery. Several samples are needed between minutes 1 to 12 to determine the time at which blood lactate levels reached a peak. In the event that a sample was missed during the 2nd through the 7th min, the test was terminated and rescheduled at a later date. If a sample was missed from the 9th through the 40th min, the point was determined to be one-half the difference between the preceding and following points (Freund & Zouloumian, 1981).

Baseline Plasma Lactate Determinations. Before the recovery tests were administered, each subject reported to the laboratory on three separate days for the determination of baseline blood lactate accumulation for each of the four treatments. The baseline value refers to the blood lactate concentration after 20 min of exercise at each of the relative workloads to be used during recovery without prior maximal exercise. The resting blood lactate value was determined during the first visit to the lab. Baselines for the 35 and 65% $\dot{V}O_2$ max were determined at 20 min of steady-state exercise because it has been shown that blood lactate accumulation does not vary significantly

during submaximal work from 20 to 60 min (Genolevy, 1980; Nagel, Robinhold, Howley, Daniels, Baptista & Stoedefalke, 1970). The baseline for the combined intensities was obtained at the end of 40 min after the subject cycled for 7 min at 65% and 33 min at 35% of $\dot{V}O_2$ max.

Instruments and Techniques of Measurement

Ventilation and Gas Exchange. Oxygen uptake was measured using open circuit spirometry. A Parkinson-Cowan dry gas meter (Model CD-4) was used to measure inspired air (V_I) through flexible tubing connected to a Rudolph valve (#2700). Expired O_2 and CO_2 fractions were sampled from a 5-liter mixing chamber by Beckman OM-11 (O_2) and LB-2 (CO_2) gas analyzers which were calibrated before each workload during the oxygen cost of cycling test and before the $\dot{V}O_2$ max test with commercially prepared gas mixtures verified by the micro-Scholander technique (Scholander, 1949).

Heart Rate. Heart rate was monitored by a Hewlett-Packard electrocardiograph (Model 1511-B) with CM-5 lead placements. An ECG strip was run the last 5 sec of each stage and a minute rate extrapolated from 4 R-R' intervals.

Blood Sampling. For resting lactate determinations, approximately 3 ml of blood were taken from a forearm vein by venepuncture without stasis with a

disposable 21-gauge needle and syringe. The blood was then transferred to a mixing tube containing 100 μ l of dried 4% sodium fluoride to inhibit glycolysis. It was then transferred to a tube containing a 7% perchloric acid solution for deproteinization. The sample was then mixed and centrifuged with the supernatant aspirated and frozen for later analysis (Appendix B).

Catherization was used for sampling blood during the recovery test as follows: A disposable needle and syringe were used to draw 2 ml of heparin (Panheparin, 100 u/ml). This was added to a vial containing 20 ml of a 0.9% sodium chloride solution. A new 20 ml disposable syringe and needle were then used to withdraw 20 ml of heparinized saline.

A tourniquet was wrapped around a forearm and the hand examined for a vein to puncture. After swabbing the area with alcohol, the catheter (Deseret Minicath-PRN Catheter, 3/4 inch, 21-gauge) was inserted about 1/2 inch into the vein. If blood could be withdrawn from the syringe, the catheterization was acceptable. The catheter was then inserted completely and secured with tape. Enough heparinized saline was then infused to clear the catheter of blood.

Thirty seconds before a sample was needed, a disposable 10-ml syringe was used to clear the catheter of the heparinized saline and approximately 1 ml of

blood. A new 10-ml syringe was then used to draw approximately 1.5 ml of blood for analysis. Heparinized saline was then reinfused to clear the catheter of blood. This procedure was followed for each sample taken. The hand was warmed throughout all tests with a heating pad.

Statistical Analysis. The study utilized a randomized block design with an analysis of variance (ANOVA) for repeated measures on blood lactate. Replications were blocked with repeated measures on blood samples taken at 15 different times. An exponential curve was fit to the mean values for each test. The portion of each curve from the 6th to the 40th min was linearized and plotted on semilogarithmic scale to determine differences in rate of removal.

CHAPTER III

Results

Mean values for the blood lactate concentrations for all experimental conditions are shown in Figure 1. Because the workloads were set relative to each subject's $\dot{V}O_2$ max, the difference in peak lactate for each of the treatments was small (<0.5 mM). The smallest peak in the mean values was seen in the 35% recovery at 10.4 mM, followed by the resting and combination recoveries at 10.8 mM and the highest concentration of 10.9 mM at the 65% recovery. Even though the ANOVA indicates that there were statistically significant ($p < 0.01$) differences in the treatments, orthogonal contrasts indicated that the differences were not at the 6th min of the recovery. Since plasma concentration of lactate is a major factor determining the rate of disappearance, it is essential for the peak lactate values for each treatment to be as close as possible to allow comparison of the four recoveries. In addition, all of the treatments reached their peak mean values at 6 min into the recovery treatment and were followed by progressive declines.

As indicated by the ANOVA (Table 2 in Appendix C), there were no statistically significant differences in

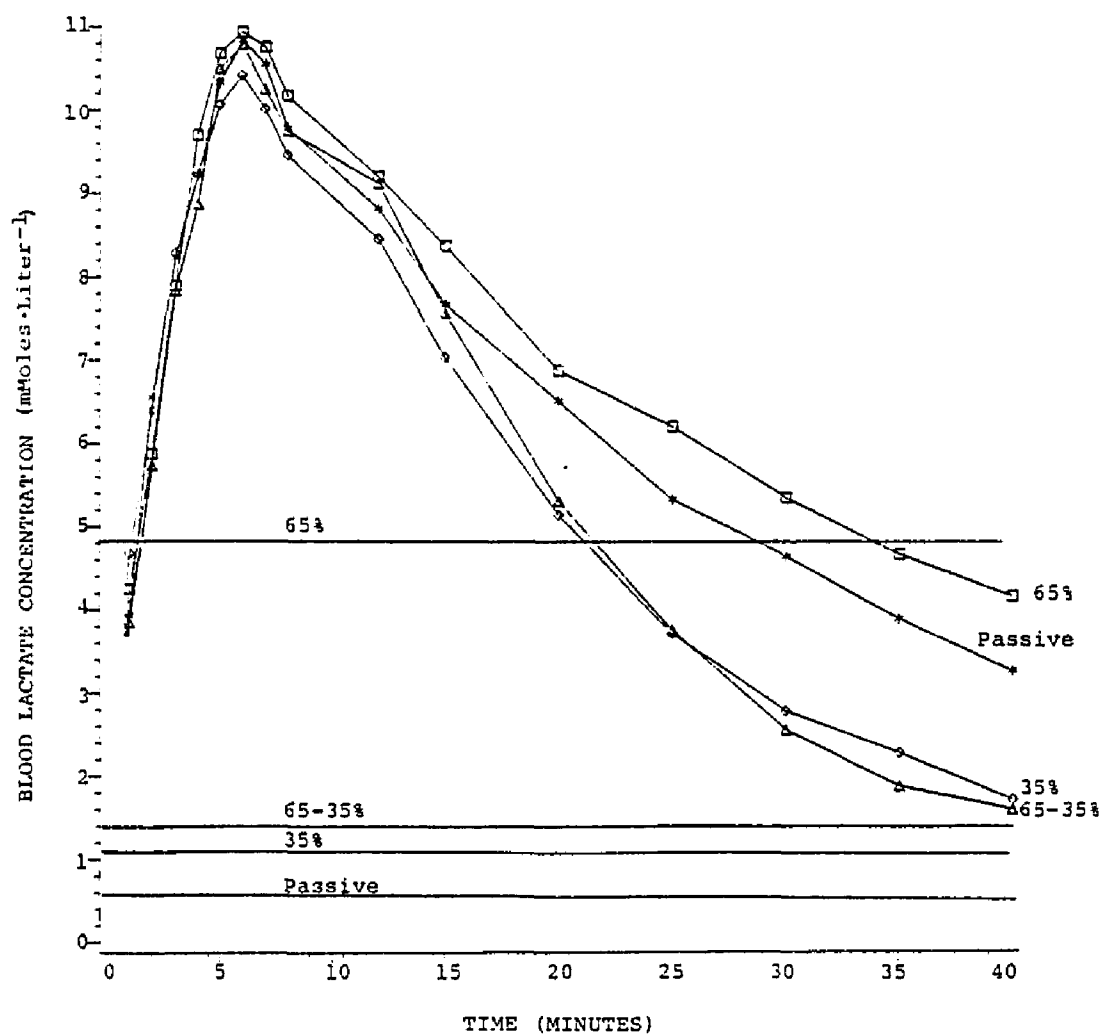


Figure 1. Mean values of blood lactate concentration for four exercise recovery intensities and predetermined baselines for each treatment.

replications of the experiment as they were set up in the design ($p > 0.05$).

Both treatments and time show statistically significant differences ($p < 0.01$) in their effects on blood lactate removal. However, in further examination, it can be seen that there is a statistically significant interaction ($p < 0.01$) between treatments and time. Thus, there is little information obtained by examining treatment or time effects separately.

Therefore, three appropriate orthogonal contrasts were selected to examine the interaction of the treatments over time. The three contrasts were: (1) 65% of $\dot{V}O_2$ max vs resting, 35% and combination 65%-35%, (2) resting recovery vs 35% $\dot{V}O_2$ max and combination 65%-35%, and (3) 35% of $\dot{V}O_2$ max vs combination 65%-35%. Each of the three contrasts were made at 6 min and from minutes 20 to 40. The comparisons revealed a statistically significant difference ($p < 0.05$) between the 65% recovery and the three other treatments from the 20th to the 40th minute. The second comparison shows a statistically significant difference ($p < 0.05$) between the resting recovery and the 35% and combination recoveries from the 20th to 40th minute. The third contrast revealed no statistically significant difference between the 35% $\dot{V}O_2$ max and combination recoveries between the 20th and 40th minute.

The blood lactate data collected at each of the recovery intensities prior to the treatments are presented as the baselines in Figure 1. The baseline at the 65% recovery was 4.8 mM and was significantly higher ($p < 0.05$) than the other treatments. The combination recovery was 1.4 mM followed by the 35% recovery at 1.1 mM and the resting recovery at 0.59 mM. There was no significant difference in the combination, 35%, or resting recoveries.

The recovery treatments can be compared to the resting baseline or to their own predetermined baseline to examine the time course of blood lactate removal. Figure 2 illustrates each treatment plotted on semi-logarithmic paper and compared to the resting blood lactate value. Comparison of curves indicates a significantly faster removal rate ($p < 0.05$) for the 35% and combination recoveries than the resting or 65% of $\dot{V}O_2$ max recoveries. These recovery values may be misleading in that some lactate could be produced during the recovery at 65% $\dot{V}O_2$ max.

Figure 3 illustrates that when the predetermined baselines were used for comparison, there were no differences in the removal rates between the three active recoveries. However, the resting recovery was significantly different than any of the active recoveries ($p < 0.05$).

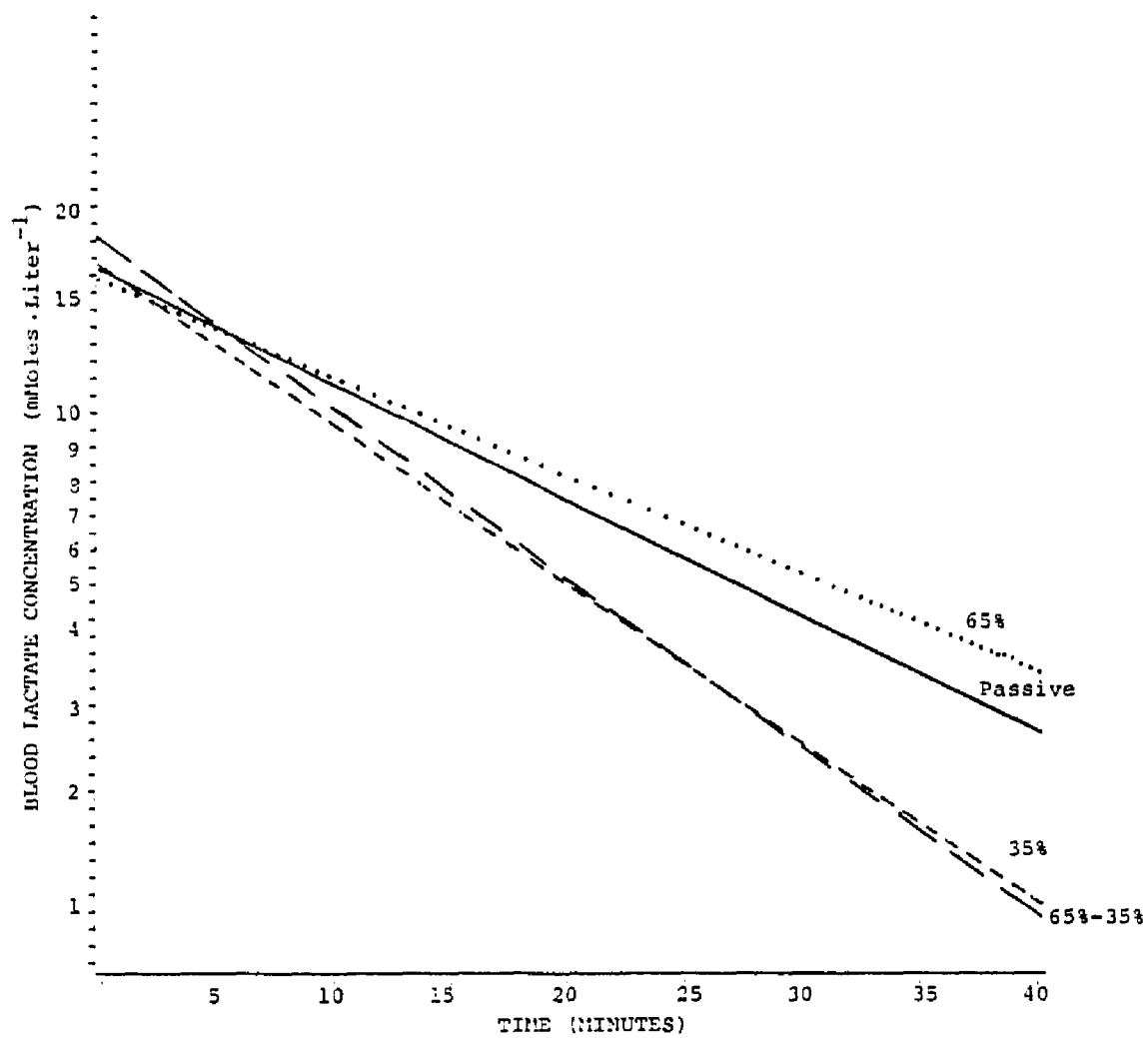


Figure 2. Semilogarithmic plot of four recovery exercise intensities when compared to resting values (Resting Mean Lactate = 0.5 mM).

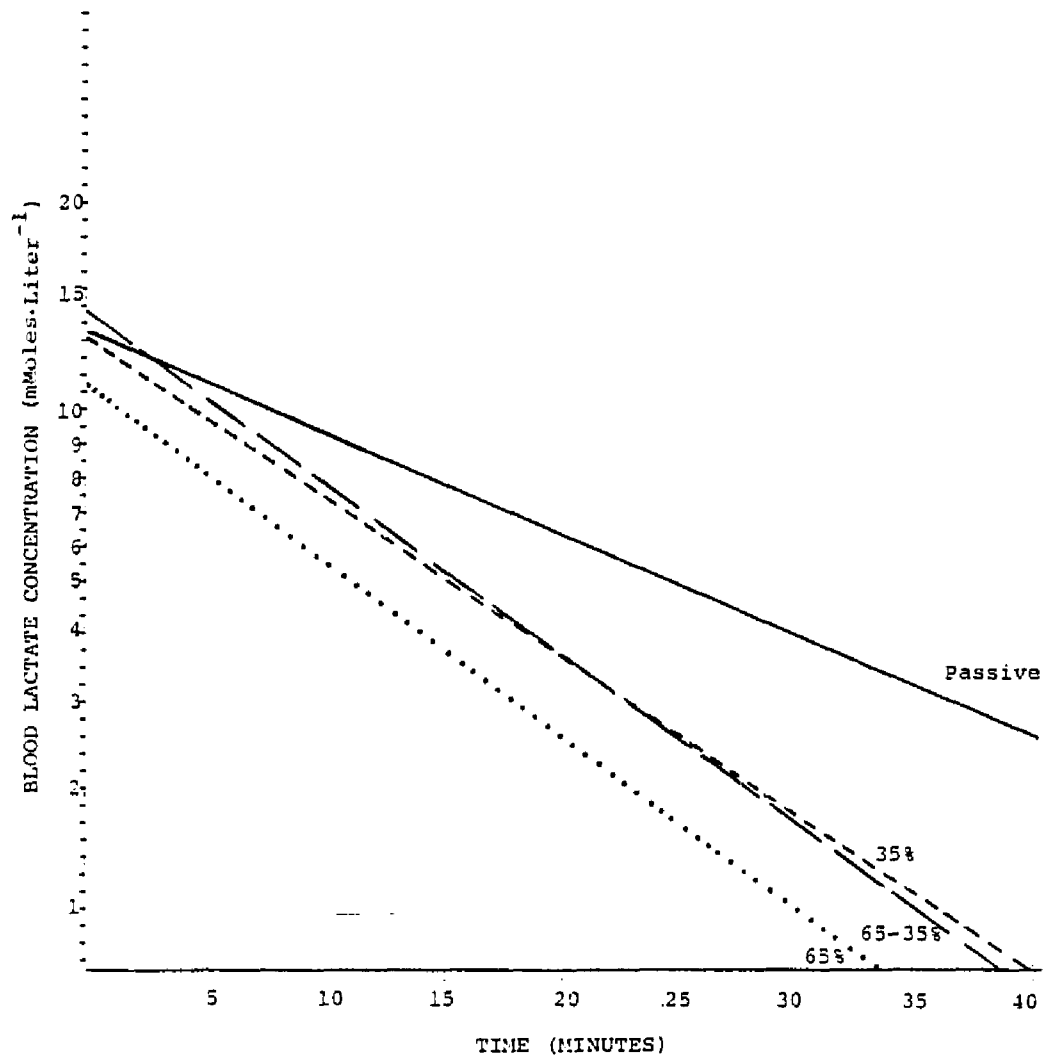


Figure 3. Semilogarithmic plot of four recovery exercise intensities when compared to predetermined baselines.

CHAPTER IV

Discussion

Valid comparisons of methods for blood lactate removal require that peak values attained by each method are similar. This prerequisite is based on the assumption that concentration is a major factor in determining the rate of removal (Bonen et al., 1979; Freund and Zouloumian, 1981). The peaks for blood lactate meet this requirement in this study. Also, the peaks all occur 6 min into the recovery which is in agreement with the time course of peak lactate values found by Margaria, Edwards and Dill (1933) and Jorfeldt (1970). In addition, it must be remembered that blood lactate concentration is an indirect "marker" of the muscle lactate concentration (Jorfeldt, et al., 1978), and thus is an indication of the qualitative changes in the muscle rather than a quantitative indication of change.

The hypothesis that the combination recovery would elicit an earlier peak in blood lactate than the other treatments was not supported by these data. Also, these data do not support the hypothesis that the combination recovery causes blood lactate to decrease at a faster rate than the other treatments. Significant differences were found in the absolute lactate values at the end of the recovery treatments; however, the hypothesis that the

combination recovery would have the lowest value was not supported. In addition, it was found that the 65% recovery had a higher absolute value than the passive recovery which is contrary to what was hypothesized.

Peak Lactate

The data of Stamford et al. (1981) suggested that peaks in blood lactate would occur earlier during recovery above the anaerobic threshold. These data do not support this finding. Both the 65% recovery and the combination recovery utilized the same workload for the first 7 min of the recovery and both indicate peak lactate at the same time with no difference in absolute values. The difference in peak blood lactate values between the study of Stamford et al. (1981) and this study may be explained by the difference in the supra-maximal work bout performed in each. Whereas this study utilized a workload corresponding to $150\% \dot{V}O_2 \text{ max}$, Stamford et al. (1981) utilized a set resistance for each subject with pedalling rate being as fast as the subject could pedal. The procedure of Stamford et al. (1981) meant that each subject performed different amounts of work in the supramaximal bout.

Removal Rate

As can be seen in Figure 2, the concentration of blood lactate in the combination recovery tended to be lower than the other three recoveries although the

difference was not significant. Blood flow may not have been higher to the working muscles during the 65% recovery. Some evidence suggests that after static work, the blood flow in the 35% recovery may have been as high as in the 65% recovery (Asmussen, 1981). Even if blood flow to the working muscle is high, if transport across the cell membrane is carrier mediated, the carrier may have been saturated at high lactate levels. Jorfeldt et al. (1978) have shown that the muscle and blood lactate levels show a linear relationship to a point of about $4 \text{ mMoles} \times \text{Kg}^{-1}$ wet weight muscle concentration after which no relationship exists. This finding suggests that the carrier responsible for the translocation of lactate is saturated at this point and no further increase in the rate of efflux of lactate can occur.

Additionally, individual characteristics of the subjects not only determine production of lactate but also the rate at which it is removed. Evans and Cureton (1982) have shown that one of these characteristics is the state of training of the individual. The state of training of the subjects in this study and that of Stamford et al. (1981) was similar when comparing the mean $\dot{V}O_2$ max of the subjects. However, Stamford's subjects may have been more specifically trained on the cycle than the subjects of this study. If this were the

case, differences between the two studies could be explained by that fact. Again, the fiber typing characteristics of the subjects may explain the differences in removal rates. This concept is supported by the data of Bonen et al. (1979) in which it was found that fiber type along with exercise intensity and lactate concentration account for differences in lactate removal. This could only account for differences found between the data of Stamford et al. (1981) and this study. Since the subjects each served as their own control in each experiment, any variation in results could not be explained by fiber type. Since no difference was found between the combination and 35% $\dot{V}O_2$ max recoveries during the first 7 min, a difference in rate of removal would not be expected thereafter since the protocols then become identical.

High Intensity Recovery

That active recovery at 65% $\dot{V}O_2$ max was less effective in removing lactate than passive recovery conflicts with Stamford et al. (1981). Whereas the data of Stamford et al. (1981) lend support to the theory that lactate may be produced only transiently, the present data suggest that lactate is produced constantly. These data also suggest that the subjects' fiber types in the two studies were different. The baseline lactate value at 70% $\dot{V}O_2$ max in the study of

Stamford et al. (1981) was 3.5 mM whereas the baseline for 65% recovery in the present study was 4.8 mM. Not only does this illustrate that the lactate turnover is greater during recovery at 65% in this study than in the 70% recovery utilized by Stamford et al. (1981), it also suggests that lactate is being produced during exercise of this intensity. Conversely, those who believe that lactate is only produced transiently may contend that the active process thought to be responsible for lactate transport across the cell membrane would explain the differences in removal rate. This is supported by the fact that blood flow is much greater to the muscle during the 65% recovery than during passive recovery. Jorfeldt (1970) presented evidence that, not only is lactate efflux from the cell inhibited at high concentrations, but lactate uptake by the muscle is also inhibited. This finding, combined with the increased production, explains the higher lactate values at 65% $\dot{V}O_2$ max recovery.

It is interesting to examine a recovery's effectiveness by comparing it to a predetermined baseline lactate value if the baseline is significantly higher than the resting baseline. The major influence of using the predetermined baseline is the effect on the rate constant found for lactate removal. Figure 3 illustrates the differences obtained in rate constants between the

treatments. It can be seen that the 65% $\dot{V}O_2$ max recovery reaches its baseline value and goes lower before the other treatments approach their individual baselines.

If the lactate concentrations were to return to the resting baseline it would be evidence that there is only transient lactate production in the muscle. However, the recovery bout was terminated before this could be determined. Also, at longer work periods the depletion of glycogen may reduce lactate production.

Practical Applications

The optimal method for removing blood lactate may be a single recovery intensity. The exact percentage of $\dot{V}O_2$ max that would be optimal for lactate removal may depend on individual characteristics of the subject (Bonen et al., 1979), but probably lies between 30-40% of $\dot{V}O_2$ max according to many investigators (Belcastro & Bonen, 1975; Gisolfi, et al., 1966). Lactate removal is probably most efficient at some point just below the anaerobic threshold; e.g., a load which creates a high rate of blood flow to working muscles but does not produce any additional lactate. Although blood flow is an important factor, it is also important to remember that metabolism plays a major role since blood lactate was lower after 40 min of recovery at 35% and higher at 65% than passive recovery.

Therefore, the competitive athlete should engage in an active recovery between events such as long sprints and middle distances in sports such as track, swimming and cycling. The intensity of the recovery would depend on the mode of exercise but is probably close to optimal if the athlete is allowed to choose the intensity (Bonen & Belcastro, 1975).

BIBLIOGRAPHY

Bibliography

- Ahlborg, G., Hagenfeldt, L., & Warren, J. Influence of lactate infusion on glucose and FFA metabolism in man. Scandinavian Journal of Clinical and Laboratory Investigation, 1976, 36, 193-201.
- Anderson, P. Capillary distribution in skeletal muscle of man. Acta Physiologica Scandinavica, 1975, 95, 203-205.
- Asmussen, E. Similarities and dissimilarities between static and dynamic exercise. Circulation Research, 1981, 48(6), 3-10.
- Asmussen, E., & Nielsen, M. Studies on the regulation of respiration in heavy work. Acta Physiologica Scandinavica, 1946, 12, 171-188.
- Astrand, P. -O., & Saltin, B. Maximal oxygen uptake and heart rate in various types of activity. Journal of Applied Physiology, 1961a, 16(6), 977-981.
- Astrand, P. -O., & Saltin, B. Oxygen uptake in the first minutes of heavy muscular exercise. Journal of Applied Physiology, 1961b, 16, 971.
- Bang, O. The lactate content of the blood during and after muscular exercise in man. Skandinavian Archives of Physiology, 1936, 74, 51-82. (Supplement 10).

- Bannister, R. G., & Cunningham, D. J. C. The effects on the respiration and performance during exercise of adding oxygen to the inspired air. Journal of Physiology, 1954, 125, 118-137.
- Bar, U., & Blanchaer, M. C. Glycogen and CO₂ production from glucose and lactate by red and white skeletal muscle. American Journal of Physiology, 1965, 209, 905-909.
- Belcastro, A. N., & Bonen, A. Lactic acid removal rates during controlled and uncontrolled recovery exercise. Journal of Applied Physiology, 1975, 39, 923-937.
- Bonen, A., & Belcastro, A. Comparison of self-selected recovery methods on lactic acid removal rates. Medicine and Science in Sports, 1976, 3(3), 176-178.
- Bonen, A., Campbell, C. J., Kirby, R. L., & Belcastro, A. N. A multiple regression model for blood lactate removal in man. Pflugers Archiv, 1979, 280, 205-210.
- Brooks, G. A., Brauner, K. E., & Cassens, R. C. Glycogen synthesis and metabolism of lactic acid after exercise. American Journal of Physiology, 1973, 224(5), 1162-1166.
- Davies, C. T. M., Knibbs, A. V., & Musgrove, J. The rate of lactic acid removal in relation to different baselines of recovery exercise. Internationale Zeitschrift fuer Angewandte Physiologie, 1970, 28, 155-161.

- Dawson, D. H., Goodfriend, T. L., & Kaplan, N. O.
Lactic dehydrogenase: functions of the two types.
Science, 1964, 143, 929-933.
- Depocas, F., Minaire, Y., & Chatonnet, J. Rates of
formation and oxidation of lactic acid in dogs at
rest and during moderate exercise. Canadian Journal
of Physiology and Pharmacology, 1969, 47, 603-610.
- Diamont, B., Karlsson, J., & Saltin, B. Muscle tissue
lactate after maximal exercise in man. Acta
Physiologica Scandinavica, 1968, 72, 383-384.
- Dieterle, P., Banholzer, P., Dieterle, R., Henner, J.,
& Schwartz, K. The influence of lactate on muscle
metabolism. Lactate as a possible regulating
factor for the increased energy supply during
muscular work. Hormone and Metabolic Research,
1971, 3, 340-344.
- Dyksta, G. L., Boileau, R. A., & Misner, J. E. Effect
of selected warm down work levels on blood lactate
removal. AAHPER Abstract Research Papers, 1973,
49. (Abstract)
- Evans, B. W. & Cureton, K. J. Effect of physical
conditioning on blood lactate disappearance after
supramaximal exercise. Medicine and Science in
Sports, 1982, 14(2), 113. (Abstract)
- Fitts, R. H., & Holloszy, J. O. Lactate and contrac-
tile force in frog muscle during development of

- fatigue and recovery. American Journal of Physiology, 1976, 231(2), 430-433.
- Freund, H. & Zouloumian, P. Lactate after exercise in man: I. Evolution kinetics in arterial blood. European Journal of Applied Physiology, 1981, 46, 121-133.
- Freyschuss, U., & Strandell, T. Limb circulation during arm and leg exercise in the supine position. Journal of Applied Physiology, 1967, 23, 163-170.
- Genolevy, H. Blood lactate concentration during prolonged submaximal exercise at varying intensities. Unpublished MS thesis, University of Louisville, 1980.
- Gisolfi, C., Robinson, S., & Turrell, E. S. Effects of aerobic work performed during recovery from exhausting work. Journal of Applied Physiology, 1966, 21, 1767-1772.
- Gollnick, P. D., & King, D. W. Energy release in the muscle cell. Medicine and Science in Sports, 1969, 1, 23-31.
- Goodale, W. T., Olson, R. E., & Hackel, D. B. The effects of fasting and diabetes mellitus on myocardial metabolism in man. American Journal of Medicine, 1959, 27, 212-220.
- Graham, T. E., & Sinclair, D. G. Metabolic intermediates and lactate diffusion in active dog skeletal

- muscle. American Journal of Physiology, 1976, 231(3), 766-771.
- Hermansen, L., & Saltin, B. Blood lactate concentration during exercise at acute exposure to altitude. In R. Margaria (Ed.), Exercise at altitude. Amsterdam, Excerpta Medica, 1967.
- Hermansen, L., & Stensvold, I. Production and removal of lactate during exercise in man. Acta Physiologica Scandinavica, 1972, 86, 191-201.
- Hill, A. V., & Lupton, H. Muscular exercise, lactic acid and the supply and utilization of oxygen. Quarterly Journal of Medicine, 1923, 16, 135-171.
- Hubbard, J. L. The effect of exercise on lactate metabolism. Journal of Physiology, London, 1973, 231, 1-18.
- Hughes, R. L., Clode, M., Edwards, R. H. T., Goodwin, T. J., & Jones, N. L. Effect of inspired O₂ on cardiopulmonary and metabolic responses to exercise in man. Journal of Applied Physiology, 1968, 24, 336-337.
- Issekutz, B., Jr., & Miller, H. I. Plasma free fatty acids during exercise and the effects of lactic acid. Proceeding of the Society of Experimental Biology and Medicine, 1962, 100, 237-243.
- Issekutz, B., Shaw, W. A. S., & Issekutz, A. C. Lactate metabolism in resting and exercising dogs.

Journal of Applied Physiology, 1976, 40, 312-319.

Jervell, O. Blood lactate removal during submaximal exercise. Acta Medica Scandinavica, 1928, 24, 1. (Supplement)

Jorfeldt, L. Metabolism of L(+)-lactate in human skeletal muscle during exercise. Acta Physiologica Scandinavica, 1970, 338. (Supplement)

Jorfeldt, L., Juhlin-Dannefelt, A., & Karlsson, J. Lactate release in relation to tissue lactate in human skeletal muscle during exercise. Journal of Applied Physiology, 1978, 44, 350-352.

Karlsson, J. Lactate in working muscle after prolonged exercise. Acta Physiologica Scandinavica, 1971a, 82, 123-130.

Karlsson, J. Lactate and phosphagen concentration in working muscle of man. With special reference to oxygen deficit at the onset of work. Acta Physiologica Scandinavica, 1971b, 358. (Supplement)

Karlsson, J., Bonde-Petersen, F., Henriksson, J., & Knuttgen, H. G. Effects of previous exercise with arms or legs on metabolism and performance in exhaustive exercise. Journal of Applied Physiology, 1975, 38, 763-767.

Keul, J., Doll, E., & Kuppler, D. The substrate supply of human skeletal muscle at rest, during and after work. Experimentia, 1967, 23, 974-979.

- Klausen, K., Knuttgen, H. G., & Forster, H. Effects of preexisting high blood lactate concentration on maximal exercise performance. Scandinavian Journal of Clinical and Laboratory Investigation, 1972, 30, 415-419.
- Krebs, H. A. Gluconeogenesis. Proceedings of the Royal Society of London, 1964, 159, 545-565.
- Kreisberg, R. A., Siegal, A. M., & Owen, W. C. Glucose-lactate interrelationships: effect of ethanol. Journal of Clinical Investigation, 1971, 50, 175-185.
- Kreisburg, R. A. Glucose-lactate inter-relations in man. The New England Journal of Medicine, 1972, July 20, 132-136.
- Margaria, R., Edwards, H. T., & Dill, D. B. The possible mechanisms of contracting and paying the oxygen debt and the role of lactic acid in muscular contraction. American Journal of Physiology, 1933, 106, 689-715.
- Nagel, F., Robinhold, D., Howley, E., Daniels, J., Baptista, G., & Stoedefalke, K. Lactic acid accumulation during running at submaximal aerobic demands. Medicine and Science in Sports, 1970, 2, 182-186.
- Newman, E. V., Dill, D. B., Edwards, H. T., & Webster, F. A. The rate of lactic acid removal in exercise.

American Journal of Physiology, 1937, 118, 457-462.

Pentecost, B. L., Reid, J. A., & Reid, D. Lactate and pyruvate metabolism in the exercising ischaemic limb. Journal of Clinical Investigation, 1966, 45, 855-864.

Poortmans, J. R., Bossche, J., & Leclercq, R. Lactate uptake by inactive forearm during progressive leg exercise. Journal of Applied Physiology: Respiratory, Environmental, and Exercise Physiology, 1978, 45, 835-839.

Reichard, G. A., Jr., Moury, N. F., Jr., & Hochella, N. J. Quantitative estimation of the Cori cycle in the human. Journal of Biological Chemistry, 1963, 238, 495-501.

Rowell, L. B. The liver as an energy source in man during exercise. In B. Pernow & B. Saltin (Eds.), Muscle metabolism during exercise. New York: Plenum, 1971.

Rowell, L. B., Blackmon, J. R., & Bruce, R. A. Indocyanine green clearance and estimated hepatic blood flow during mild to maximal exercise in upright man. Journal of Clinical Investigation, 1964, 43, 1677-1690.

Rowell, L. B., Kraning, K. K., Evans, T. O., Kennedy, K. W., Blackmon, J. R., & Kusumi, F. Splanchnic

- removal of lactate and pyruvate during prolonged exercise in man. Journal of Applied Physiology, 1966, 21, 1773-1783.
- Saiki, H., Margaria, R., & Cuttica, F. Lactic acid production in submaximal work. Internationale Zeitschrift fuer Angewandte Physiologie, 1967, 24, 57-61.
- Scholander, P. F. Analyzer for accurate estimation of respiration gases in one-half cubic centimeter samples. Journal of Biological Chemistry, 1947, 167, 235-250.
- Sjodin, B. Lactate dehydrogenase in human skeletal muscle. Acta Physiologica Scandinavica, 1976, 436. (Supplement)
- Stainsby, W. N., & Welch, H. G. Lactate metabolism of contracting dog skeletal muscle in situ. American Journal of Physiology, 1966, 211, 177-183.
- Stamford, B. A., Moffatt, R. J., Weltman, A., Maldonado, C., & Curtis, M. Blood lactate disappearance following supramaximal one-legged exercise. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology, 1978, 45, 244-248.
- Stamford, B. A., Rowland, R., & Moffatt, R. J. Effects of severe prior exercise on assessment of maximal oxygen uptake. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology, 1978, 44, 559-563.

- Stamford, B. A., Weltman, A., Moffatt, R., & Sady, S.
Exercise recovery above and below anaerobic threshold following maximal work. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology, 1981, 51(4), 840-844.
- Stenberg, J., Astrand, P. -O., Ekblom, B., Royce, J., & Saltin, B. Hemodynamic response to work with different muscle groups, sitting and supine. Journal of Applied Physiology, 1967, 22, 61-70.
- Stryer, L. Biochemistry. San Francisco: W. H. Freeman & So., 2nd ed., 1981, pp. 352-354.
- Waterhouse, C., & Keilson, J. Cori cycle activity in man. Journal of Clinical Investigation, 1969, 48, 2359-2366.
- Welch, H. B., Bonde-Peterson, F., Graham, T., Klausen, K., & Secher, N. Effects of hyperoxia on leg blood flow and metabolism during exercise. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology, 1977, 42, 385-390.
- Weltman, A., Stamford, B. A., & Fulco, C. Recovery from maximal effort exercise: lactate disappearance and subsequent performance. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology, 1979, 47, 677-682.

APPENDICES

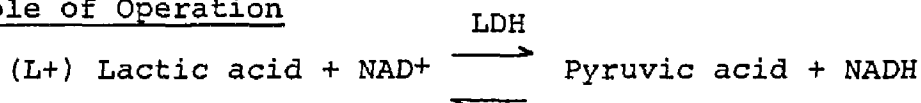
Appendix ASubject Consent Form

My signature on this sheet, by which I volunteer to participate in the experiment on the effects of active recovery on blood lactate levels after supramaximal exercise, conducted by Steve Dodd, indicates that I understand and agree to the following:

1. That I have or will be informed of the nature of the experiment.
2. That each test will involve venous catheterization to be performed by certified medical personnel.
3. That I will not eat within 4 hours of any of the tests.
4. That I will be in a rested state having done no strenuous exercise within 12 hours of any of the tests.
5. All subjects in the project are volunteers.
6. That I can withdraw at any time from the experiment.
7. That the data I provide will be anonymous and my identity will not be revealed without my permission.
8. That I shall be given an opportunity to ask questions prior to the start of the experiment and after my participation is complete.

Date

Subject's Signature

Appendix BEnzymatic Lactic Acid AnalysisPrinciple of Operation

In the presence of LDH, lactic acid and NAD⁺ are converted to pyruvic acid and NADH, respectively. The amount of NAD⁺ which is converted to NADH is measured spectrophotometrically @ 340 nm and becomes a measurement of the lactate originally present.

Even though excess NAD⁺ and LDH are present, the reaction will only go to completion if the pyruvic acid is removed. Therefore, a hydrazine-glycine buffer is added to remove the pyruvate end product. Since lactic acid is the only molecule that will readily participate in this particular reaction, it is extremely reliable for that substance.

Reagents

1. 70% perchloric acid (HClO₄)
2. Hydrazine-Glycine Buffer
Hydrazine sulfate -- Sigma No. H-3376
Glycine -- Sigma No. G-7126
3. Lactate Dehydrogenase (LDH) -- Sigma No. L-2625
4. Nicotinamide Adenine Dinucleotide, Ox. Form --
Sigma No. N-8129

5. Lithium (L+) Lactate Standard-Stock Sigma (L+)
Lithium Lactate (L2250)
20 mM = 38.4 mg brought to 10 ml with H₂O
6. 4% NaF (w/v)

Daily Work Solutions

1. 7% Perchloric Acid (100 ml)
Add 6.0 ml of 70% HClO₄ to a flask and bring to 100 ml with H₂O (Keep refrigerated)
2. Hydrazine-Glycine Buffer (100 ml)
 - (a) 0.4 M Hydrazine and 0.5 M Glycine. Add 3.76 gm Glycine and 5.20 gm Hydrazine Sulfate to about 80 ml H₂O
 - (b) Adjust pH to 9.0 with concentrated NaOH and bring to 100 ml with H₂O
 - (c) Stable for 1 week
 - (d) Start a cocktail with 1 ml buffer/sample to be measured.
- *3. Lactic Dehydrogenase
Add enough LDH to the cocktail so that the final concentration is 25 μ l/sample.
- *4. Nicotinamide Adenine Dinucleotide, Ox. Form.
Add enough NAD to the cocktail that the final concentration is 1.25 mg/sample.
- *5. Add enough H₂O to the cocktail so that the final concentration is 2 ml/sample.

*Should be added to cocktail only before use (i.e., after all samples and standards have been pipetted).

6. Working Standards

Add H_2O to all volumes of stock to make correct serial dilutions.

2mM = 0.5 ml stock up to 10 ml

6mM = 1.5 ml stock up to 10 ml

10mM = 2.5 ml stock up to 10 ml

15mM = 3.75 ml stock up to 10 ml

7. Sample Collection

(a) 0.05 ml of 4% NaF is added to 12 x 75 mm test tube and dried overnight at 80°C.

(b) 1.5 ml of a blood sample is added to the NaF tube and mixed.

(c) 1.0 ml of the whole blood is then added to a tube containing 2 ml of 7% perchloric acid for deproteinization. This is mixed and either refrigerated or centrifuged.

(d) After the sample is centrifuged, the supernatant is drawn off to a separate tube and frozen until analysis.

(e) Standard Curve. Add 100 μ l of each working standard to 200 μ l of cold 7% $HClO_4$. Mix thoroughly and treat as a blood sample.

(f) Analytical Procedure

(1) Standards and samples should be analyzed in duplicate. Add 100 μ l of Blank, Standards and Samples to test tube.

(2) Treat all of the above tubes as follows:

- a. Add 1.5 ml buffer to each tube.
- b. Mix thoroughly.
- c. Incubate at 37°C for 30 min
and cool to room temperature for
15 min.

(g) Absorbance Determination

- (1) Read absorbance at 340 nm
- (2) Use perchlorate blank to correct all readings.

(h) Calculations

- (1) Due to quenching of the spectrophotometer, the curve is not linear. To compensate, a standard curve must be used to get correct concentrations.

Appendix C

Table 2

Repeated measures ANOVA for blood lactate removal
during four recovery treatments.

| Source | df | Sum of Squares | F | Prob F |
|---|-----|----------------|--------|--------|
| Replications | 3 | 36.37 | 2.24 | .1229 |
| Treatment | 3 | 90.05 | 5.55 | .0083 |
| Replication * Treatment | 9 | 433.61 | 8.91 | .0001 |
| Error (a) Subject (Replication * Treatment) | 16 | 86.54 | 3.75 | .0001 |
| Time | 15 | 4865.26 | 224.82 | .0001 |
| Treatment * Time | 45 | 116.53 | 1.79 | .0018 |
| Error (b) | 420 | 605.95 | | |
| Total | 511 | 6234.31 | | |

Vita

Stephen Lollar Dodd was born in Vallejo, California, on December 11, 1949. Soon after, his family moved to Birmingham, Alabama, where he grew up and graduated from Erwin High School in May, 1968.

He received a Bachelor of Science degree in Economics from the University of Alabama in 1973 after serving one year in the U.S. Navy from 1971 to 1972. He then completed requirements for a Bachelor of Science degree in Physical Education at the University of Alabama-Birmingham in 1974. After completion, he accepted a teaching-coaching position at Woodlawn High School in Birmingham, which he held until May of 1976.

He then returned to the University of Alabama-Birmingham to receive his Master of Arts in Physical Education in 1978. He was then offered a position as an instructor in physical education at the University of Alabama-Birmingham which he accepted. He taught until resigning in August of 1979 to enter Louisiana State University to work on the Ph.D. degree in physical education. His major emphasis of study was in exercise physiology with a minor in zoology. He accepted a position as assistant professor in the Department of Health, Physical Education and Recreation at Mississippi State University in August of 1982 and was graduated at Louisiana State University in December of 1982.

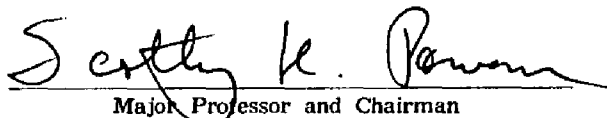
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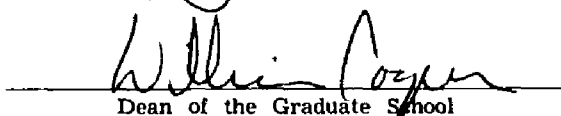
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Major Field: Physical Education

Title of Thesis: BLOOD LACTATE REMOVAL DURING VARYING
INTENSITIES OF ACTIVE RECOVERY
FOLLOWING SUPRAMAXIMAL WORK

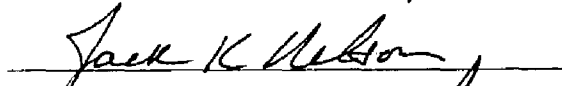
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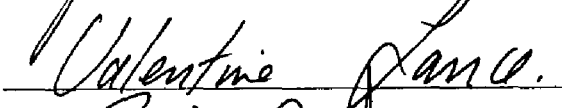

Major Professor and Chairman

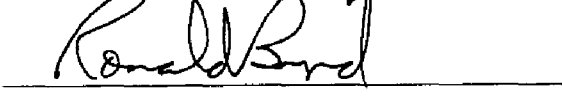

Dean of the Graduate School


EXAMINING COMMITTEE:











Date of Examination:

November 29, 1982